ENCLOSURE 7

IN THE UNITED STATES COURT OF APPEALS

FOR THE NINTH CIRCUIT

No. 91-70389

LONGVIEW FIBRE CO., et al.,

Petitioners,

v.

DANA A. RASMUSSEN, Regional Administrator, and the UNITED STATES ENVIRONMENTAL PROTECTION AGENCY,

Respondents.

No. 91-70398

COLUMBIA RIVER UNITED, et al.,

Petitioners,

v.

DANA A. RASMUSSEN, Regional Administrator, and the UNITED STATES ENVIRONMENTAL PROTECTION AGENCY,

Respondents.

REPLY DECLARATION OF IAN CHRISTOPHER NISBET

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I, IAN CHRISTOPHER THOMAS NISBET, declare as follows:

1. My name is Ian Christopher Thomas Nisbet. I made a declaration in this case on November 11, 1991. In that declaration, I expressed the professional opinion that the Total Maximum Daily Loading (TMDL) of 2,3,7,8-TCDD promulgated by the U.S. Environmental Protection Agency (EPA) for the Columbia River Basin was scientifically deficient and would not protect wildlife, including bald eagles, living along the river, from toxic effects of 2,3,7,8-TCDD and related chemicals.

2. On February 18, 1992, EPA submitted a brief in this case, to which was attached a declaration by Steven P. Bradbury, dated February 12, 1992. Dr. Bradbury's declaration contested some of the points made in my first declaration and concluded that the TMDL would, in fact, protect bald eagles against toxic effects of 2,3,7,8-TCDD. This second declaration responds to certain points made in Dr. Bradbury's declaration and in EPA's brief. Paragraphs 3-10 herein point out and restate some points made in my first declaration that were not contested by Dr. Bradbury. Paragraphs 11-21 herein respond to statements made by Dr. Bradbury. Paragraphs 22-24 herein respond to other issues concerning my first declaration, raised in EPA's brief. Paragraph 25 herein restates my opinion concerning threats to wildlife, including bald eagles.

3. Paragraph 9 in my first declaration stated that the TMDL was based on EPA's Ambient Water Quality Criterion (AWQC) for 2,3,7,8-TCDD, and that this AWQC was promulgated without any consideration of wildlife. This point was not addressed or

otherwise contested by Dr. Bradbury.

4. Paragraph 11 in my first declaration stated that the AWQC Document was issued in 1984 and was out of date. This point was not contested by Dr. Bradbury, and was not addressed except by his reference (his paragraph 4) to an ongoing effort by EPA to develop water quality criteria for 2,3,7,8-TCDD that would protect wildlife in the Great Lakes Basin.

5. Paragraph 12 in my first declaration stated that the treatment in the 1984 AWQC Document of the bioconcentration of 2,3,7,8-TCDD in fish was seriously deficient, and that the bioconcentration factor (BCF) of 5,000 used in the AWQC Document was much too small. This point was acknowledged by Dr. Bradbury, who recommended and used (his paragraphs 8c and 8d) a BCF of 90,000. Thus, based on Dr. Bradbury's recommended value for the BCF, the AWQC Document of 1984, and the TMDL Document of 1991, would have underestimated the exposure of fish-eating animals, including humans and wildlife, by a factor of 18.

6. Paragraph 13 in my first declaration pointed out that the TMDL Document failed to take account of the existing contamination of the Columbia River with 2,3,7,8-TCDD in calculating the permissible daily loading. Specifically, I referred to Appendix B in the TMDL Document, which discussed the exchange of 2,3,7,8-TCDD between water and sediments, resulting in prolonged retention of discharged 2,3,7,8-TCDD in sediments. Although Dr. Bradbury addressed this point in his paragraph 11b, he misunderstood the basis for my argument (see comments in paragraph 21 below). He did not contest my primary point, that

the TMDL Document did not consider the facts set out in Appendix B when calculating the TMDL from the AWQC.

7. Paragraph 14 in my first declaration pointed out that the TMDL established a maximum daily loading only for 2,3,7,8-TCDD, and not for other chemicals in the families known as polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs), some of which are known to occur along with 2,3,7,8-TCDD in pulp mill discharges. I also pointed out that in so doing, EPA had failed to apply its own procedures for calculating the toxicity of mixtures of PCDDs and PCDFs -- viz., the use of Toxic Equivalency Factors or TEFs. The arguments in Dr. Bradbury's declaration were limited to 2,3,7,8-TCDD and did not address other PCDDs, PCDFs, or TEFs in any way. Thus, Dr. Bradbury did not contest my statement that the TMDL Document had understated the risks to consumers of fish and had overstated the degree of protection.

8. Paragraph 15 in my first declaration pointed out that bald eagles in the Columbia River Basin were already exposed to 2,3,7,8-TCDD and other related chemicals, that they had accumulated high levels of some of these chemicals in their tissues, and that their reproduction was already impaired as a result of these exposures. Although Dr. Bradbury addressed the issue of incremental exposure to 2,3,7,8-TCDD in his paragraph 11b, he merely presented an argument that exposure of bald eagles to 2,3,7,8-TCDD would decrease as a result of implementing the TMDL (see further comments in paragraph 21 below). He did not address in any way my statement that current exposure to 2,3,7,8-

TCDD was already contributing to reproductive impairment; nor did he consider any chemicals other than 2,3,7,8-TCDD. Thus, my arguments in these respects were uncontested.

9. Paragraph 8 in my first declaration stated that many of the toxic effects of 2,3,7,8-TCDD and other dioxin-like compounds have been observed in populations of wild mammals or birds. Paragraph 10 stated that some wildlife species, including the mink and certain fish-eating birds, are especially susceptible to the toxic effects of PCDDs. Paragraph 15 stated that exposure of fish-eating birds in the Columbia River Basin is expected to lead to tissue residues of 2,3,7,8-TCDD that are well into the ranges associated with toxic effects in other species. For this reason, I considered it probable that 2,3,7,8-TCDD at existing levels of contamination is already contributing to reproductive impairment in bald eagles in the Columbia River Basin. These statements were supported by citations to published scientific literature, including five papers reporting effects on wild birds. Dr. Bradbury's declaration did not contest these statements; indeed, he relied entirely on experimental studies of captive birds (pheasants and chickens) in the laboratory, and did not cite or otherwise consider any studies in wild mammals or birds. In contrast, I relied in my declaration on both experimental studies and field studies; I believe this to be a more scientific approach.

10. In summary, the statements in my first declaration that are referred to in paragraphs 3-9 above remain essentially uncontested. These statements remain my professional opinions

and are hereby restated.

11. Dr. Bradbury refers in paragraph 4 of his declaration to his participation in an ongoing effort by EPA and certain state agencies to develop water quality criteria for the protection of wildlife in the Great Lakes Basin. This effort is currently incomplete and both the methodology for developing criteria and the specific criteria values will be subject to internal review, peer-review, public comments and revision before they will be finalized or adopted as EPA policy. The only output from this effort that he cites is an internal draft document, dated November 1991, concerning proposed procedures for determining bioaccumulation factors. Dr. Bradbury does not cite any other outputs from this program as the basis for his scientific opinions. Accordingly, I will limit my responses in the following paragraphs to his statements in paragraphs 8-11 of his declaration.

12. The primary studies on which Dr. Bradbury bases his calculations of the risks posed to bald eagles by water containing 0.013 ppq of 2,3,7,8-TCDD are those referenced in his bibliography as authored by U.S. EPA (1991), Nosek (1991), Cook et al. (1991) and Cook (1992). All of these documents were completed after February 1991, i.e., after the issuance of the TMDL. Thus, they could not have been the basis for the TMDL or for any conclusion that the TMDL would protect wildlife. Dr. Bradbury's arguments represent, at best, a post hoc rationalization for the TMDL.

13. Dr. Bradbury's calculations consider only 2,3,7,8-TCDD.

Thus, even if they were completely unobjectionable, they would merely provide an estimate of the concentration of 2,3,7,8-TCDD in ambient water that would protect bald eagles in the absence of exposure to any other toxic compounds. In the Columbia River Basin, as pointed out in my declaration (paragraphs 13-15), bald eagles are exposed to other toxic contaminants, including other PCDDs, PCDFs, and PCBs. Some of these contaminants are known to act by the same mechanisms as 2,3,7,8-TCDD and to augment its effects in animals exposed simultaneously. Hence, the level of exposure to 2,3,7,8-TCDD that the Columbia River bald eagles could tolerate without adverse effects will be less than it would be in the absence of the other contaminants. Dr. Bradbury's approach, therefore, overestimates the level of exposure that these birds could tolerate. A specific calculation, using the TEF approach or another scientifically valid approach, would have to be performed before Dr. Bradbury's conclusion could be applied to bald eagles in the Columbia River Basin.

14. Dr. Bradbury bases his calculations of safety on an unpublished doctoral dissertation by Nosek (1991), which he cites as reporting a "no effect level" of 2,3,7,8-TCDD in ring-necked pheasants. Although Dr. Bradbury states that the results of Nosek's study have been accepted for publication in a scientific journal, they have not yet been published and hence are not available for review. In my opinion, the route and schedule of exposure in Nosek's study (injection of 2,3,7,8-TCDD into the body cavity once weekly for 10 weeks) raise questions that need review. In particular, measurements of levels of 2,3,7,8-TCDD in

the eggs would be needed to establish the doses administered to the embryos.

15. Dr. Bradbury states (footnote to paragraph 8h) that the chicken is approximately 3 fold more sensitive [to PCBs] than the ring-necked pheasant, and that it is therefore "appropriately conservative" to assume that the [bald] eagle may be up to 10 times more sensitive [to 2,3,7,8-TCDD] than the ring-necked pheasant. On the basis of the information cited, this statement is highly speculative. The studies cited for the proposition that the pheasant were conducted in different laboratories and by different methods. Based on the most comparable data -- reduction in hatchability of eggs laid by females exposed orally to Aroclor 1254 -- I calculate that the chickens were about <u>six</u> times more sensitive than the pheasants, not three times. However, the toxic effects of Aroclor 1254 (a commercial mixture of PCBs) are not exclusively dioxin-like, and these results do not provide a sound basis for estimating the relative sensitivity of chickens and pheasants to 2,3,7,8-TCDD. Even if they did, no basis is given for the assumption that a further factor of 3.3 (or any other factor) would provide sufficient protection for bald eagles.

16. A much sounder basis for estimating the relative sensitivity of pheasants and chickens to 2,3,7,8-TCDD is provided by the experiments of Brunstrom and his colleagues [1, 2]. These investigators studied pheasants and chickens in the same laboratory by the same methods. They injected various doses of 3,3',4,4'-tetrachlorobiphenyl (TCB) into fertile eggs and

observed effects on hatchability. TCB is one of the most dioxin-like components of PCB mixtures, is known to act by the same mechanism as 2,3,7,8-TCDD, and should be a good surrogate for the effects of 2,3,7,8-TCDD [2]. The results of Brunstrom and his colleagues (summarized in Table 1 of ref. 2) indicate that chicken embryos are about fifty times more sensitive to TCB than are pheasant embryos. Thus, Dr. Bradbury's factor of 10 for relative sensitivity would not protect chickens, let alone bald eagles.

17. Dr. Bradbury did not cite a study by Cheung et al. (ref. 24 in my first declaration, ref. 3 in this declaration), even though this study has been used by EPA as the basis for earlier wildlife risk assessments. This study provides a direct measure of the sensitivity of chicken embryos to 2,3,7,8-TCDD, by-passing the necessity to rely on data from pheasants and speculations about relative sensitivity.

18. Dr. Bradbury based his assessment of exposure and risks to bald eagles on the assumption that bald eagles eat only fish. He considered it "reasonable" to do so (paragraph 11a), in spite of citing data that indicate that bald eagles actually eat variable quantities of other prey, including fish-eating birds. For the Columbia River Basin, direct information on the diet of bald eagles is provided by the report of Garrett et al. [4], cited both by myself (ref. 9 in my first declaration) and by Dr. Bradbury. The authors of this report actually reported wide variations in the diets of bald eagles in the Columbia River Basin, with major seasonal and pair-to-pair variations (pp. 66-

74). Birds comprised 27 percent of prey found at nests in May and June, 7 percent of prey captured by nesting eagles, and 28 percent of prey capture by wintering eagles (pp. 63, 67). These figures represent the proportions of birds in the diet by frequency; the proportions by weight were much higher, because the birds taken were generally much larger than the fish. Based on the data presented in the report, the average eagle in the study would have consumed fish-eating birds as at least 10 percent of the diet by weight during the breeding season, and at least 25 percent of the diet by weight during the months prior to egg-laying.

19. Dr. Bradbury evidently considered that if birds comprised only between 3 and 10 percent of the diet, their contribution to the eagles' exposure to 2,3,7,8-TCDD would be negligible (his paragraph 11 b). This is a serious error, however. As I pointed out in my first declaration (paragraph 15, citing to refs. 11 and 14), fish-eating birds concentrate 2,3,7,8-TCDD in their tissues to levels on the order of 30-40 times higher than those in the fish they eat. Thus, even if fish-eating birds comprise only 3 percent of the eagles' diet, they would contribute more 2,3,7,8-TCDD to the eagles' total intake than they 97 percent of the diet consisting of fish.

20. For reasons stated in paragraphs 11-19, foregoing, the arguments and citations presented by Dr. Bradbury do not support his conclusion that an ambient water concentration of 0.013 ppq of 2,3,7,8-TCDD would not be hazardous to bald eagles, even in the absence of pre-existing contamination and exposure to other

contaminants.

21. In his paragraph 11b, Dr. Bradbury contests my statement (paragraph 15 in my first declaration) that continued release of 2,3,7,8-TCDD would augment exposure of the eagles. However, Dr. Bradbury misstated -- and apparently misunderstood -- my argument. I neither stated nor implied, as Dr. Bradbury states, that a reduction in the concentration of 2,3,7,8-TCDD in water would result in an increase in exposure to eagles. I referred to my paragraph 13, in which I discussed (based on Appendix B in the TMDL Document) the retention of discharged 2,3,7,8-TCDD in sediments, followed by uptake into fish. Continued discharge of 2,3,7,8-TCDD will augment the total quantity of 2,3,7,8-TCDD in the system and will, at least in the short term, increase the exposure of fish, fish-eating birds, and bald eagles. On a long time-scale, some of the 2,3,7,8-TCDD will be transported downstream on resuspended sediments. Changes in the quantity and distribution of 2,3,7,8-TCDD in the system, and in the concentrations of 2,3,7,8-TCDD in sediments, water, fish, and fish-eating birds, could be calculated using models incorporating the considerations outlined in Appendix B of the TMDL Document. Such calculations would probably show that the concentrations of 2,3,7,8-TCDD will continue to increase in some parts of the system, even after the rate of discharge is reduced, although they may decrease in other parts of the system (e.g., stretches of the river where contaminated sediments have not accumulated). However, no calculations of this kind were performed by EPA in the text of the TMDL Document, so neither EPA

nor Dr. Bradbury has any basis for asserting that the TMDL will be protective. In particular, it is not self-evident, as assumed by Dr. Bradbury, that a decrease in discharges into the system will result in a decrease in concentrations in water and in exposure to eagles. Likewise, the assumption of EPA that the concentration of 2,3,7,8-TCDD in water in the Columbia River will be equal to the TMDL divided by the volume flow in the river is inconsistent with the information presented in Appendix B, and is simply wrong.

22. Respondent's Brief (at p. 38) cites a 1986 Biological Report issued by the U.S. Fish & Wildlife Service as "provid[ing] that '2,3,7,8-TCDD concentrations ... should not exceed ... 10 to 12 ppt in food items of birds and other wildlife.'" The document cited actually states (at p. 28) that "[d]iets containing up to 10 to 12 ppt of 2,3,7,8-TCDD may prove to be non-hazardous to birds and other wildlife, as judged by the results of laboratory studies with rats, monkeys, and chickens, and by the recommendations of New York State for human health protection." (Emphasis added.) In any case, this document issued in 1986 did not cite most of the studies regarded as important by myself and by Dr. Bradbury, and its tentative conclusions are now obsolete.

23. Respondent's brief (at pp. 39-40) cited an earlier (1990) risk assessment by EPA which indicates "that adverse effects to wildlife could potentially occur if there were greater than 3 ppt dioxin in their diet." This conclusion was not cited in the TMDL Document, or by Dr. Bradbury. In fact, recent data [5] for the Columbia River Basin show concentrations of 2,3,7,8-

TCDD in the range of 2.9-16 ppt in Caspian terns, 3.6-49 ppt in double-crested cormorants, up to 1 ppt in ring-billed gulls, and 0.7-23 ppt in western gulls, as well as 1-9 ppt in various fish. I understand that the data reported for birds are the concentrations of 2,3,7,8-TCDD in whole eggs. In most birds, concentrations in whole eggs are similar to those in whole bodies, so the ranges given would be approximately those in the corresponding components of the diet of bald eagles. Thus, on EPA's own assessment, even birds that eat exclusively fish would be at risk from 2,3,7,8-TCDD alone. The bald eagle, with up to 25 percent fish-eating birds in the diet in the period prior to egg-laying (see paragraph 18 above) is clearly at high risk. Peregrine falcons would be at still higher risk.

24. Respondent's brief (at p. 65) contests my conclusion (paragraph 15 in my first declaration) that there is "good reason" to conclude that Columbia River eagles are presently exposed to hazardous levels of 2,3,7,8-TCDD. The brief states, first, that my conclusion was based "upon his studies of <u>other</u> contaminants in <u>other</u> areas..." (emphases in original). This statement is false. My conclusion was immediately explained in the succeeding five sentences, which cited only information on 2,3,7,8-TCDD, including one specific study on contamination in Columbia River fish. The brief then stated that my conclusion was not based on "any scientific information about the level of dioxin that is known to be harmful to bald eagles." This statement is disingenuous at best. As Dr. Bradbury himself points out (his paragraph 8e), there are no direct studies of

toxic effects of 2,3,7,8-TCDD in bald eagles, because this is a species protected under the Endangered Species Act and could not be used for experimental toxicity studies. My conclusion, rather, was based on reasonable scientific inference from studies in other species. Finally, my prediction of the level of exposure of Columbia River bald eagles to 2,3,7,8-TCDD has been fully confirmed by recent data on residue levels in two eagle eggs [5]. My "good reason" has now been strengthened: it is now fully documented that Columbia River bald eagles are currently exposed to levels of 2,3,7,8-TCDD that are associated with adverse effects in other species.

25. On the basis of the foregoing considerations, I maintain and restate the professional opinions expressed in my first declaration, including the following:

a. The TMDL was not based on any consideration of wildlife protection and would not protect wildlife, including bald eagles, in the Columbia River Basin.

b. The TMDL did not take into account the known properties of 2,3,7,8-TCDD, including the dynamic partitioning between sediments, water, and biota described in Appendix B to the TMDL Document.

c. Bald eagles in the Columbia River Basin are already suffering from impaired reproduction, attributable to exposure to a mixture of chlorinated hydrocarbons, including 2,3,7,8-TCDD.

d. The current exposure of bald eagles in the Columbia River Basin to 2,3,7,8-TCDD is sufficient to cause or contribute to impaired reproduction.

e. Continued discharge of 2,3,7,8-TCDD at rates permitted by the TMDL into the Columbia River system will augment the exposure of bald eagles and increase the severity of adverse effects.

I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge. Executed this 28th day of February, 1992, in Lincoln, Massachusetts.

Ian Christopher Thomas Nisbet

Ian C. T. Nisbet

28 February 1992

402NISB3.DEC

REFERENCES

[References cited in my declaration dated November 11, 1991, are incorporated herein by reference.]

1. BRUNSTROM, B. and P.O. DARNERUD. 1983. Toxicity and distribution in chick embryos of 3,3',4,4'-tetrachlorobiphenyl injected into the eggs. Toxicology, 27: 103-110.

2. BRUNSTROM, B. and L. REUTERGARDH. 1986. Differences in sensitivity of some avian species to the embryotoxicity of a PCB, 3,3',4,4'-tetrachlorobiphenyl, injected into the eggs. Environmental Pollution, A 42: 37-45.

3. CHEUNG, M.O., E.F. GILBERT, and R.E. PETERSON. 1981. Cardiovasculararteratogenicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin in the chick embryo. Toxicology and Applied Pharmacology, 61: 197-204.

4. GARRETT, M., R.G. ANTHONY, J.W. WATSON, and K. MCGARIGAL. 1988. Ecology of Bald Eagles on the Lower Columbia River. Unpublished report to U.S. Army Corps of Engineers. 189 pp.

5. U.S. FISH AND WILDLIFE SERVICE. 1991. Organochlorine summary. Data printout, December 16, 1991. U.S. Fish and Wildlife Service, Portland Field Office. Portland, OR. 14 pp.

CERTIFICATE OF SERVICE

I am a citizen of the United States and a resident of the county of King. I am over 18 years of age and not a party to this action. My business address is 705 Second Ave., Suite 203, Seattle, Washington, 98104.

On March 3, 1992, I served a true copy of REPLY BRIEF OF PETITIONER CRU ET AL. on the persons listed below by placing said copy in a sealed envelope with postage full prepaid, in a United States Postal Service mail box in Seattle, Washington, addressed as follows:

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I, Lisa H. Lange, declare under penalty of perjury that the foregoing is true and correct.

Executed on this 3rd day of March, 1992, at Seattle, Washington.

Lisa H. Lange